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The Effect of Antiseptic Sprays on the Bacterial Content of Air

The Lessening Menace of Benzol Poisoning in American Industry

Lead Poisoning from Lead Piped Water Supplies

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THE EFFECT OF ANTISEPTIC SPRAYS ON THE BACTERIAL CONTENT OF AIR*

S. R. DOUGLAS, LEONARD HILL, AND WILSON SMITH

From the National Institute for Medical Research, Hampstead, London

EXPERIMENTS WITH ANTISEPTIC SPRAYS

THE organism chosen for the experiments was *Bacillus coli communis*, a nonpathogenic and easily cultivated organism, the colonies of which growing on MacConkey's agar plates are readily identified and counted.

Technic.—Horse muscle digest broth (30 c.c.) was sown with this organism and incubated at 37°C. for approximately eighteen hours; the culture was then used to make a homogeneous emulsion (1 part of culture to 4 parts of 0.85 per cent. saline). A measured quantity of this emulsion was sprayed from a spraying bottle with a very fine nozzle into a room 5,208 cu. ft. in capacity, the windows and doors of which were closed; the spray was directed over the exit of a powerful electric fan, the blast of which was vertically upward. The result was a wide dispersion of bacteria in the atmosphere of the room. The average time taken to spray 5 c.c. of emulsion was sixty seconds. Ten minutes after the onset of bacterial spraying, MacConkey's agar plates were exposed for a period of two minutes.

In the first two series of experiments, plates of approximately the same size were grouped in three different positions in the room, three plates in each group for each exposure. Plates of the same group were found to exhibit approximately the same number of colonies; therefore only one of each group was counted and the average of the three counts recorded. Later, we used four plates disposed in pairs, the first pair on a table, marked *A*, the second pair on a bench higher than the table, marked *B*. In most cases the *A* counts were found to be somewhat

* Received for publication Feb. 9, 1928.

higher than the *B* counts. After exposure the plates were incubated overnight at 37°C. and the lactose fermenting colonies counted the following morning.

In experiments where the effect of an antiseptic was to be tested this was sprayed from a cheap but effective spray, "The Sovereign," for two periods of one minute each in the interval between bacterial dissemination and plate exposure. The operator walked slowly round the room spraying at the rate of about one stroke of the piston per second and directing the spray in all directions except downward. The total volume of fluid thus sprayed in the two periods was about 80 c.c.

At the conclusion of each experiment the room was thoroughly cleared by opening windows and doors wide and running the electric fan. An analysis of the protocols shows that this clearance was quite effective.

The procedure was as follows:

Start of experiment: Fan on with bacterial spray.
End of spray: Fan off. (Approximately 1 minute from start.)
Interval of 2 minutes. | Or Interval of 9 minutes in the dry experiments.
First antiseptic spray (for 1 minute).
Interval of 2 minutes.
Second antiseptic spray (for 1 minute).
Interval of 3 minutes.
Plates exposed for 2 minutes.
Room thoroughly cleared.

The antiseptic sprays used were a well-known commercial preparation containing terpenes with oxidizing properties, hereafter indicated as *S*, and a well-known commercial preparation of electrolized sea water with about 1 per cent. of available chlorine, hereafter indicated as *M*.

TABLE 1.—RESULTS OF SPRAY EXPERIMENTS: SERIES 1 AND 2

EXPERIMENT NO.	C.C. OF 1/5 BACTERIAL EMULSION	ANTISEPTIC	COLONIES	
			Average No.	Per Cent.[1]
Series 1				
1	5	dry	489	103.0
2	5	*S* (1/5)	471	99.2
3	5	dry	460	96.9
Series 2				
1	5	dry	315	119.3
2	5	*S* (1/5)	95	35.9
3	5	dry	213	80.6
4	5	water	180	68.1

[1] Mean of dry counts taken as 100 in Tables 1 to 6, inclusive.

Series 1.—This series comprises three experiments, the first and third being "dry," *i.e.*, without any antiseptic spraying between bacterial dissemination and plate exposure, the second being to test the effect of *S* (1 in 5 dilution in tap water). Three plates were exposed in different parts of the room and the mean of the three counts was recorded. The details and results are shown in Table 1.

Series 2.—This was a repetition of the first series except that a fourth experiment was added to test the effect of a spray of tap water. The results shown in Table 1, compared with those of Series 1, are somewhat more significant. It will be seen that the *S* count is only 36 per cent. of the

mean dry count, while tap water gave a count of 68 per cent.

Series 3.—Six experiments were included in this series, two dry, two water, one *S*, and one *M*. Four plates were used in each, set out in pairs as described above, and a record of each separate count was kept.

It will be seen from Table 2 that all the counts are remarkably low, as compared with those of the preceding series; the technic, however, was precisely the same, as was also the quantity of organisms sprayed. We suggest as the probable factor excessive dryness of surface of the MacConkey plates.

Series 4.—An experiment was designed to find out whether the antiseptic acts on the bacteria suspended in the air, or on the culture medium, rendering the plates unsuitable for colony formation.

S (1 in 4 dilution) was sprayed into the room for one minute; after an interval of two minutes this spraying was repeated. After a further two-minute interval two plates marked *A1* and *B1* were exposed in the usual manner. The room was then thoroughly cleared.

The bacterial spraying was now performed, using 7.5 c.c. of 1 in 5 emulsion (7.5 c.c. were used instead of the usual 5 c.c. because of the exceptionally low counts in the experiments of Series 3). After an interval of three and one-half minutes, plates *A1* and *B1* were reexposed together with two fresh plates marked *A2* and *B2*.

The counts were too high to be made accurately. All four plates, however, had between 500 and 600 colonies, so that no inhibition by preliminary exposure of the culture medium to the air of a room sprayed with *S* could be detected.

TABLE 2.—RESULTS OF SPRAY EXPERIMENTS: SERIES 3

EXPERIMENT NO.	C.C. OF 1/5 BACTERIAL EMULSION	ANTISEPTIC	COLONIES					
			No. on Each Plate				Average No.	Per Cent.
			A1	A2	B1	B2		
1	5	dry	4	3	2	2	2.75	61.1
2	5	*S* (1/4)	0	0	0	1	0.25	5.5
3	5	water	18	20	12	22	18.00	400.0
4	5	dry	10	8	4	3	6.25	138.8
5	5	*M* (1/5)	8	7	3	4	5.50	122.2
6	5	water	6	14	3	7	7.50	166.6

TABLE 3.—RESULTS OF SPRAY EXPERIMENTS: SERIES 5

EXPERIMENT NO.	C.C. OF 1/5 BACTERIAL EMULSION	ANTISEPTIC	COLONIES	
			Average No.	Per Cent.
1	7.5	dry	479.5	103.5
2	7.5	*S* (1/4)	325.0	70.1
3	7.5	water	419.5	90.6
4	7.5	dry	446.5	96.4

Series 5.—This was a repetition of Series 2, except that 7.5 c.c. of 1 in 5 bacterial emulsion were sprayed instead of 5 c.c. The results, which again suggest a distinct effect on the part of *S*, are shown in Table 3.

Series 6.—The details of this series are sufficiently shown in Table 4. A remarkable effect was obtained with *M;* none whatever with *S*. In this case a freshly opened bottle of *M* was used.

Series 7.—A repetition of the last series in which we reverted to the use

of 5 c.c. of bacterial emulsion gave similar results, which are also shown in Table 4.

TABLE 4.—RESULTS OF SPRAY EXPERIMENTS: SERIES 6 AND 7

EXPERIMENT NO.	C.C. OF 1/5 BACTERIAL EMULSION	ANTISEPTIC	COLONIES					
			No. on Each Plate				Average No.	Per Cent.
			A1	A2	B1	B2		
Series 6								
1	6	dry	306	307	285	258	289.0	96.1
2	6	*S* (1/5)	339	346	383	348	354.0	117.7
3	6	*M* (1/4)	23	14	10	7	13.5	4.4
4	6	water	279	237	298	282	274.0	91.1
5	6	dry	297	293	339	321	312.5	103.9
6	6	*S* (1/5)	232	277	351	346	301.5	100.2
Series 7								
1	5	dry	323	317	264	298	300.5	101.9
2	5	*S* (1/5)	291	308	270	251	280.0	95.0
3	5	water	260	291	253	210	253.5	86.0
4	5	*M* (1/4)	18	15	10	11	13.5	4.5
5	5	dry	334	317	260	245	289.0	98.0
6	5	*S* (1/5)	302	244	255	258	264.7	89.8

Series 8.—The inefficiency of *S* in the two preceding series was thought possibly to be due to the fact that the same bottle of disinfectant had been used throughout, so that by this time it might have deteriorated. The experiments were therefore repeated using freshly purchased bottles of both *S* and *M*.

In Experiments 1 to 4, 5 c.c. of 1 in 5 bacterial emulsion were used. In Experiments 5 and 6 the effect of *M* and *S*, respectively, was tested after a 7.5-c.c. spray of 1 in 5 bacterial emulsion. The results presented in Table 5 show that both antiseptics exert an effect, that of *M* being of quite remarkable potency. They also confirmed our suspicion that the antiseptics become less potent after the bottles have been opened for a certain period.

TABLE 5.—RESULTS OF SPRAY EXPERIMENTS: SERIES 8

EXPERIMENT NO.	C.C. OF 1/5 BACTERIAL EMULSION	ANTISEPTIC	COLONIES					
			No. on Each Plate				Average No.	Per Cent.
			A1	A2	B1	B2		
1	5	dry	50	39	39	37	41.2	148.7
2	5	*S* (1/5)	10	8	9	14	10.2	36.8
3	5	*M* (1/4)	0	0	0	0	0.0	0.0
4	5	dry	17	18	8	14	14.2	51.2
5	7.5	*M* (1/4)	0	0	0	0	0.0	.....
6	7.5	*S* (1/5)	12	19	14	14	14.7	.....

TABLE 6.—RESULTS OF SPRAY EXPERIMENTS: SERIES 9 AND 10

EXPERIMENT NO.	C.C. OF 1/5 BACTERIAL EMULSION	ANTISEPTIC	COLONIES					
			No. on Each Plate				Average No.	Per Cent.
			A1	A2	B1	B2		
Series 9								
1	5	dry	439	450	405	..[1]	431.3	100.0
2	5	*M* (1/4)	220	225	195	203	210.7	48.8
3	5	*M* (1/24)	330	300	342	332	326.0	75.5
4	5	*M* (1/48)	296	325	403	..[1]	341.3	79.1
Series 10								
1	5	dry	106	187	146	134	143.2	98.5
2	5	*M* (1/4)	2	1	3	1	1.7	1.2
3	5	*M* (1/24)	44	59	43	62	52.0	35.7
4	5	dry	160	174	112	143	147.5	101.5

[1] Plate broken.

Series 9.—This series of experiments was devised in order to ascertain whether *M* used as a spray in much higher dilution has any appreciable bactericidal effect. This was found to be the case, and the results also show a rough parallelism between bactericidal effect and degree of dilution (see Table 6). It should be noted that the bottle of *M* used in this series had been opened a few days previously.

Series 10.—This series was designed to confirm the results of Series 9. Only two dilutions of *M* were tested but two dry controls were included, one at the beginning of the series, the other at the end. The more striking bactericidal effect in this series was probably due to the fact that a freshly opened bottle of *M* was used. (See Table 6.)

Experiments with Antiseptics in Solution

Experiments were carried out with *S* and *M* in solution in order to estimate their power of inhibiting the growth of *Bacillus coli* in broth, and also to determine whether their effect on bacterial emulsions *in vitro* offers any parallelism to the effects demonstrated in the spray experiments.

Effect on Bacillus Coli in Nutrient Broth

Experiment 1.—Dilutions of *M* were made, using sterile distilled water as diluent, and were added to tubes of horse muscle digest broth so that each tube contained 7 c.c. of fluid and the final dilutions of the antiseptic were 1 in 7, 1 in 70, 1 in 700, and 1 in 7,000. A control tube was included to which sterile water was added in place of *M*. All the tubes were seeded by means of a capillary pipette with an equal quantity of an eight-hour broth culture of *Bacillus coli communis*, the same strain of organism being used as in the spray experiments. Tubes were incubated for twenty-four hours at 37°C.

The results were as follows:

Dilution of M	*Growth*[1]
1/7	−
1/70	+
1/700	+
1/7,000	+
Control	+

Experiment 2.—This was a repetition of Experiment 1, except that different dilutions of *M* were used:

Dilution of M	*Growth*
1/10	−
1/20	+−
1/30	+
1/40	+
1/50	+
1/60	+
1/70	+
Control	+

Experiment 3.—*S* was tested in a similar way. The dilutions used and the results were as follows:

Dilution of S	*Growth*
1/5	−
1/10	−
1/20	−
1/40	−
1/80	+
1/160	+
1/320	+
Control	+

Experiment 4.—The same technic was employed as in the preceding experiments, but *M* and *S* were tested

[1] In this and subsequent tabulations, − = no growth; + = profuse growth; +− = slight growth.

side by side and in the same dilutions. New bottles of the disinfectants were obtained for the experiment.

	Growth	
Dilution	*S*	*M*
1/10	−	−
1/20	−	+−
1/30	−	+
1/40	−	+
1/50	−	+
1/60	−	+
1/70	−	+
1/80	+	+
Control	+	+

In Experiments 1 to 4 the effect of *S* was much greater than that of *M*, a reversal of the order established in the spray experiments. It was thought, however, that this failure on the part of *M* might be due to a fixation of its active constituent by protein constituents of the broth. The following experiment therefore was devised.

Effect on Emulsions of Bacillus Coli

Experiment 5.—An emulsion of *Bacillus coli communis* was made in the same manner as in the spray experiments using twenty-four-hour broth culture and adding 1 part of culture to 4 parts of 0.85 per cent. saline. This emulsion was distributed to sterile tubes, arranged in two rows, in quantities of 1 c.c. to each tube. To each tube of the front row, 1 c.c. of a dilution of *S* in sterile distilled water was added so that the final dilutions of *S* were 1 in 2, 1 in 4, 1 in 8, 1 in 16, 1 in 32. *M* was added in exactly the same way to the tubes of the back row.

The mixtures were allowed to stand for fifteen minutes at room temperature, being shaken from time to time. One loopful of each mixture was then plated out on MacConkey's agar. The plates were incubated at 37°C. for twenty-four hours with the following results:

	Growth	
Dilution	*S*	*M*
1/2	+	−
1/4	+	−
1/8	+	−
1/16	+	−
1/32	+	−

Experiment 6.—This was merely a repetition of Experiment 5 using freshly opened bottles of the disinfectants and carrying the dilutions further:

	Growth	
Dilution	*S*	*M*
1/2	+	−
1/4	+	−
1/8	+	−
1/16	+	−
1/32	+	−
1/64	+	−
1/128	+	+

Experiment 7.—For this experiment 1 c.c. of the following dilutions of *S* was added to sterile tubes:

Tube	*Dilution*
1	1/1
2	1/1
3	1/2
4	1/4
5	1/8
6	1/16

To each tube, 1 c.c. of a 1 in 5 *Bacillus coli* emulsion was added. The mixtures were thoroughly shaken at intervals. After fifteen minutes a loopful of each of the mixtures in tubes 2 and 3 was plated out on MacConkey's agar. Immediately afterward 2 c.c. of broth were added to each tube. Tube 1 was immersed in boiling water for fifteen minutes to kill all the organisms for subsequent use as an opacity standard,

the other tubes were incubated at 37°C. for twenty-four hours. The *S* dilutions were now 1 in 4, 1 in 8, 1 in 16, 1 in 32, and 1 in 64. The results were as follows:

Dilution of S	*Growth*
1/4	−
1/8	−
1/16	+
1/32	++
1/64	++

The tubes marked − showed no greater opacity than the heated control tube; those marked + showed much greater opacity. Both the MacConkey plates showed numerous colonies.

Loopfuls of the mixtures in those tubes which showed no evidence of growth after their twenty-four-hour incubation (1 in 4 and 1 in 8 *S* dilutions) were again plated out on MacConkey's agar. No colonies were obtained. This indicates that prolonged action of *S* in these dilutions not merely inhibits growth but actually kills the organisms.

Observations carried out by one of us (L. H.) on the ciliated epithelium of the trachea of the horse showed that 1 per cent. *M* and 10 per cent. *S* in physiologic saline sufficed to lessen or to stop the action of the cilia. The action on cilia therefore agrees with that on *Bacillus coli*.

Summary and Discussion

1. A method is described for estimating the bactericidal power of an antiseptic spray.

2. Under the conditions of the experiments the two antiseptic sprays tested, *M* and *S*, were shown to exert a definite bactericidal effect, with *M* much the greater.

3. The bactericidal effect of an antiseptic spray depends upon many factors, among which are: (*a*) the antiseptic used; (*b*) the degree of dilution; and (*c*) the age, exposure, etc., of the antiseptic.

4. In the case of the two antiseptics used in the experiments a parallelism is demonstrated between their effects on *Bacillus coli* when used as sprays, and when mixed as solutions with a mass emulsion of organisms. This parallelism does not hold when they are used to inhibit bacterial growth in a fluid medium, such as nutrient broth; in this case the order of potency is reversed.

While from the foregoing it would appear that the use of an antiseptic spray in crowded workrooms, offices, theaters, etc., is justified, it must be borne in mind that the experimental conditions—namely, massive pollution of the atmosphere in a room, with all windows and doors closed, with a single strain of a nonpathogenic microorganism—differ greatly from those obtaining in ordinary, or even overcrowded, buildings. The effect of the antiseptics used in the sprays might be different on a mixture of organisms derived from the occupants. Again, in these experiments the dose of antiseptic used was very large and the use of a similar quantity in industrial life would be objectionable. On the other hand, there would be far fewer microorganisms present in the air, even in crowded rooms.

It will be observed that the dry counts of different series show wide variation, even when the same quantity of bacterial emulsion was sprayed.

The dry counts, however, of any one series approximate each other very closely. We have already advanced the suggestion that the degree of dryness of the plates is one factor in this variation. Furthermore, from day to day, atmospheric conditions play a part no doubt by influencing the natural ventilation of the room. The effect of ventilation in diminishing the number of micro-organisms was shown in a previous paper.[2]

Although the use of strong antiseptic sprays might be objectionable to some individuals, we found that compared with water sprays they were refreshing.

[2] Douglas, S. R., and Hill, L.: Spray Infection. THIS JOUR., 1921–1922, *3*, 206.

The experiments in which *M* and *S* were used in solution prove *M* to be a powerful and rapid germicide when brought into contact with a saline suspension of *Bacillus coli*, but comparatively inert when used in a fluid such as nutrient broth. *S*, on the other hand, is not so efficacious in the former case, but exhibits greater germicidal power in the latter. This difference might possibly render an *S* spray more effective than an *M* spray, should the air borne bacteria be surrounded by a protein containing material, such as dried feces or sputum.

CONCLUSION

Antiseptic sprays exert a bactericidal effect on *Bacillus coli communis* suspended in the atmosphere.

THE LESSENING MENACE OF BENZOL POISONING IN AMERICAN INDUSTRY*

ALICE HAMILTON, M.D.

IN THE issue of *The Journal of the American Medical Association* for March 4, 1922, there appeared an article over my name entitled "The Growing Menace of Benzene (Benzol) Poisoning in American Industry." This embodied the results of an inquiry into the industrial use of benzol at the time, made with the assistance of manufacturers and insurance companies. Only six years have elapsed since that date, but so great have been the changes in manufacturing processes during this short period that I am impelled to write a brief report on the now lessening danger from benzol in industry. Were I to wait another year it is probable that the showing would be even more favorable.

The credit for this great and beneficent change should be given primarily to the Chemical and Rubber Sections of the National Safety Council, an association of industrialists and companies writing industrial insurance, which at the annual meeting in Detroit in the fall of 1922 appointed a committee[1] to study and report on the use of benzol in industry and its possible harmful effects. The work of this Committee was prolonged for three years and in May, 1926, a final report was issued (1) (2) in which were described the chief industrial uses of benzol, the methods of use, and the incidence of benzol poisoning among workers as determined by clinical symptoms together with examination of the blood. A quantitative estimation of benzol in the air of factories was made, and by checking up the blood picture of those examined with the air analysis it was possible to determine the lowest concentration of benzol in the air which might bring about a leukopenia. It was found that 32 per cent. of the workers examined had leukopenia, mild or pronounced, and that this occurred in workrooms with good exhaust ventilation and low atmospheric contamination, as low as 100 parts of benzol per million of air. The Committee was thus "forced to conclude that the control of the benzol hazard (except where the substance is used in completely closed systems) is exceedingly difficult; that in practice, systems of exhaust ven-

* Received for publication April 9, 1928.

[1] The Committee was constituted as follows: C.-E. A. Winslow, Yale, Chairman; Leonard Greenburg, United States Public Health Service, Vice Chairman; W. S. Paine, Aetna Life Insurance Company, Secretary; H. Bradshaw, E. I. du Pont de Nemours & Company; J. W. S. Brady, M.D., Massachusetts General Hospital; A. C. Fieldner, United States Bureau of Mines; C. F. Horan, Hood Rubber Company; L. E. Weber, Consulting Chemist; J. M. Weiss, Manufacturing Chemists' Association.

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J. I. H.
Sept., 1928

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[1] The Committee was constituted as follows: C.-E. A. Winslow, Yale, Chairman; Leonard Greenburg, United States Public Health Service, Vice Chairman; W. S. Paine, Aetna Life Insurance Company, Secretary; H. Bradshaw, E. I. du Pont de Nemours & Company; J. W. S. Brady, M.D., Massachusetts General Hospital; A. C. Fieldner, United States Bureau of Mines; C. F. Horan, Hood Rubber Company; L. E. Weber, Consulting Chemist; J. M. Weiss, Manufacturing Chemists' Association.

tilation capable of keeping the concentration of benzol in the atmosphere below 100 parts per million are extremely rare; and that, even when this is accomplished, there remains a decreased, but substantial, hazard of benzol poisoning." They recommended the substitution of toluol and xylol for benzol, and described experiments carried out at Yale University by J. J. Batchelor (3) which showed that these homologues of benzol are far less toxic than benzol itself, producing only a slight anemia and having no effect on the leukocytes (4).

Even before the publication of the Committee's final report, the changed attitude of industry toward the use of benzol had become evident. A large shoe company which had used benzol in cement wrote me that because of its experience with one girl employee who had excessive and irregular menstruation it had abandoned the use of benzol although the new solvent involved the expenditure of $150 a day more than the old. Machine shops spraying coatings began to use lacquer made with toluol. Many rubber factories returned to the use of petroleum solvents for spread rubber goods and for most of their cements. In dry cleaning, benzol was already disappearing, and the protests of the organized painters against the use of this thinner in quick-drying paints were beginning to have some effect.

It was, however, the introduction of rubber latex (5) as a substitute for rubber dissolved in benzol that made possible the greatest changes in the benzol using industries. Latex is the milky fluid which is drawn off from the rubber tree after slitting the bark. The practice in rubber plantations has always been to coagulate it at once, make it into sheets, and ship it in this form, but by this new method coagulation is prevented by the addition of ammonia, which inhibits the growth of coagulating bacteria, and the latex is shipped to this country practically unchanged. Various substances (secret) are added to it to make it plastic, viscous, waterproof, and rustproof and to color it. This liquid rubber is finding extensive use in industries which formerly required benzol-rubber solutions, or naphtha-rubber, especially for impregnating fabrics, or for cementing, or for producing hermetically sealed containers.

Present Use of Benzol in Industry

The National Safety Council listed as the chief benzol using industries in 1922: (*a*) rubber, especially tire building; (*b*) artificial leather manufacture; (*c*) sanitary can manufacture; (*d*) dry cleaning; (*e*) paints, varnishes, and stains—their use rather than their production. Let us take these industries in order and see what the situation is at the present day with regard to the use of benzol.

Rubber Industries

Rubber tire building still employs benzol as a solvent for the cement, and large amounts are used. The risk is much greater for the tire builder than for the cement mixer; in fact, when mixing is properly carried on there is no escape of benzol vapor. But in tire building each layer of rubberized cord or cloth is painted over with benzol cement, and the tire is built up of many layers. The nature of the

work does not permit placing exhaust ventilation in such a position as to carry off all the dangerous fumes. It was in tire building that Harrington's (6) five cases of benzol poisoning, with aplastic anemia and hemorrhages, occurred, all in men, all severe, and three fatal. Although benzol has not been displaced in tire building, there are other processes in tire making where latex is being introduced. Thus cord fabric is now made by drawing parallel cords through a bath of latex and drying them carefully in the proper spacing so that a sheet of fabric results in which the cords are held together by a wall of rubber. It is said also that inner tubes can be made from latex.

Benzol is still used in making rubber cement for shoes and for fastening rubber heels on leather shoes, as can be seen from a recent article by Hunter and Hanflig (7) describing six cases of benzol poisoning, three of them in shoe factory workers, and a fourth in a cobbler, all of whom used a cement containing a large proportion of benzol. Two were women, one of whom died; one of the men also died. Many shoe factories, however, have given up benzol cement and are using latex, for leather, for rubber and leather, and for fastening sole welting.

In making spread rubber goods, impregnating fabric by pressing rubber dough into it and driving off the solvent by heat, benzol may be used, although apparently not often as a regular procedure; still it may be used from time to time as the market for the petroleum solvents varies and as benzol is for the time being more available and cheaper than naphtha. Making spread rubber goods is a process which produces almost invariably dangerous quantities of benzol vapor, as the rubber dough is spread by means of a long, fixed knife over the fabric which then passes on over steam pipes, to emerge from under a hood or out from a chamber still warm and with some of the benzol still evaporating (8). That the escape of fumes is inevitable is seen from the high fire insurance rates imposed in such a plant. Even when naphtha is used, the work is unhealthful, and it is therefore a great advance in industrial hygiene to be able to substitute latex for rubber solution in making spread rubber, as one of the largest firms in the country is already doing.

Dipped rubber goods, such as surgical gloves, may now be made from latex instead of rubber-benzol, as I have myself seen done, and we are assured that this use of latex will be greatly extended in the future.

Artificial Leather Manufacture

Artificial leather manufacture involves the use of great quantities of coating which, up to the present time, has always contained benzol.[2] In reading the reports of benzol poisoning in the literature it is often difficult to determine whether the victim was occupied in making artificial leather, fabrikoid, by spreading a coating on fabric, or in making patent leather by spreading a coating on skins. Essentially the two processes are the same, for in both the coating contains benzol. These new coatings, which are also used now as lacquers for all sorts of surfaces, consist of nitrocellulose dis-

[2] According to Greenburg of the National Safety Council Committee, there may be as much as 60 per cent. of benzol.

solved in normal butyl acetate or sometimes a mixture of ethyl, amyl, and butyl acetates, together with normal butyl alcohol. Some oil, usually castor oil, is added as a plasticizer, and then benzol or toluol is added as thinner and solvent. Normal butyl acetate would do the work alone, but it is much more expensive than the aromatics and therefore they are used up to the point at which they cease to act as solvents and begin to precipitate the nitro cotton. At present lacquers used for spraying usually contain toluol, but for patent leather and artificial leather the more quickly evaporating benzol is preferred by many manufacturers, although some have given up benzol and are now using toluol because of its greater safety.

The coating may be spread over the fabric in the same way that rubber is spread, or it may be applied by a stiff brush, or pressed into the leather with a broad knife held slantingly. Necessarily, in doing this hand work, the men must bend over the skin or fabric, and it is difficult to see how exhaust ventilation could be so placed as to protect them efficiently, for it is even difficult to provide adequate window ventilation without causing drafts of air which might deposit dust on the coating and spoil it. This is especially true of patent leather where the slightest blemish shows.

Two of Hunter and Hanflig's (7) six cases contracted benzol poisoning in making patent leather, and in a personal communication from New Jersey (1927) I was informed of a fatal case in a man doing the same work.

Sanitary Can Manufacture

Perhaps the most notorious source of benzol poisoning in American industry has been the manufacture of so-called sanitary cans for food products. Selling's (9) famous cases came from a Maryland factory making cans; so did McClure's (10); so did Hogan and Shrader's (11); and two which were reported to me personally in 1925 came, one from Maryland, the other from the Pacific coast. Insurance men have assured me that for every case of benzol poisoning in the can industry which came to light there were at least ten which remained unpublished. The fact that Maryland factories so often occupied the limelight was due not to worse conditions in these plants but to the brilliant work of Selling of the Johns Hopkins Hospital, which early called the attention of Baltimore to this condition.

These "sanitary cans" began some twenty years ago to take the place of the former lead-soldered cans as hermetically sealed containers for "processed goods," *i.e.*, cooked and sterilized foods. They have several advantages over the old type of can which required skilled solderers, whom it is difficult to find in the remote country districts where most canneries are situated. Moreover, the opening in the old type of can was at the most only 2 inches in diameter, and such vegetables as tomatoes and sweet potatoes had to be crushed to get them in. The new can is made with ends which are the full diameter of the can, so that filling is greatly facilitated. Sealing is effected not by solder but by clamping the end around the body of the can. Something, however,

must be used to make it absolutely air-tight. This can be done by a paper gasket if the contents of the can are somewhat liquid, for then the paper swells and makes the joint tight. I saw paper gaskets in use in the great Dole pineapple cannery at Honolulu. They can also be used in canning tomatoes and corn, but not for drier foods such as spinach, beans, fish, nor for liquids. For these it has been customary to use a thin rubber cement made with benzol, a very fine stream of which is forced into the channel around the edge of the end piece, the benzol being then driven off in a heated chamber and the end piece coming out with a thin band of the rubber in place. Since it is easier for canneries to use one kind of can for all their products, there is not nearly so much demand for paper gaskets as for rubber-sealed cans, and the can making industry was, up to two years ago, one of the largest consumers of benzol in the country. It was among the women and girls who received the can ends from the evaporating oven and inspected and stacked them while still warm that the cases of benzol poisoning developed.[3] As recently as March, 1928, an article appeared in THIS JOURNAL, by Adelaide Ross Smith (13), describing benzol poisoning in women employed at such work, in three New York can factories. Among twenty-five women exposed to benzol fumes, six showed evidence of chronic benzol poisoning.

During the last eighteen months this industry seems to have undergone a revolutionary change and benzol has been largely eliminated. This was brought about certainly in part by the earnest desire of some manufacturers to put a stop to the continually recurring cases of poisoning among the young women employed as takers-off, cases which appeared even in the best ventilated factories. The new sealing compound is rubber latex, and the rapid development of its use may be seen in the statement of a firm which produces sealing mixtures. Formerly this firm made for the trade about 500,000 gallons of benzol-rubber compound a year; now they make hardly 12,000, all the rest of their production being latex.

Recently it was possible for me to visit five large can factories, belonging to three companies. In all but one of these, latex is used for sealing cans. The largest company in the country has adopted it altogether, the next largest has introduced it only partially, still using in some branches a rubber solution, but not benzol. In visiting these places it was easy to see that contamination of the air with benzol fumes must have been inescapable even in the best factory, while in one where I found fifty-four coating machines closely crowded together in one rather low-ceiled room, the situation must have been fraught with real danger. It is hardly an exaggeration to say that this change in sealing mixture means as great a reform in the large and growing can producing industry as was brought about in the match industry by the substitution of sesquisulphide of phosphorus for yellow phosphorus. The hermetic sealing of bottles, however, is still

[3] The case of fatal benzol poisoning reported by Speidel and Melgard (12) was in a woman who is said to have dipped rubber rings in benzol and then applied them to the ends of the cans.

effected by means of a rubber-benzol mixture.

Dry Cleaning Industry

Dry cleaners do not use benzol any more in New England. As to the rest of the country, I cannot speak with positiveness. I have a personal communication concerning a woman severely poisoned in a dyeing and cleaning establishment in Indiana, in 1927, but that is the only instance which has come to my knowledge in recent years. The fire hazard from benzol is great, leading to high insurance rates, and naphtha is generally used in its place. The naphtha selected has of late years been one of the higher-boiling and less volatile distillates, because the lower-boiling fractions are needed for motor car fuel. A still less volatile distillate, known as "Stoddard's Solvent" which distils between naphtha and kerosene, is beginning to make its way into dry cleaning establishments, and, since it is classed by the insurance companies after evaporation tests as no greater a risk than kerosene, it is evidently much less volatile and therefore less toxic than naphtha.

Paint Industries

The fifth important industry in the National Safety Council's list is the painting trade, where benzol is added as a solvent and a thinner to paints, varnishes, and stains, and is an important constituent of paint and varnish removers. Benzol evaporates much more quickly than naphtha or even petroleum ether, and therefore is valued as a thinner for quick-drying paints and for nitrocellulose lacquers. In this field also, recent years have seen a change for the better, for much of the lacquer now used for spray coating is made with toluol, and there is a strong effort on the part of painters' organizations in such states as New York, Massachusetts, New Jersey, and Wisconsin to prohibit the use of benzol in any coatings which are to be applied by spraying.

Haircloth Manufacture

Another industry which deserves mention although it is not so important as the others is the making of haircloth. Formerly a solution of rubber in benzol or naphtha was used to bind the material together and prevent the hairs from pulling out; now it is found that latex can be used for this purpose very successfully.

Summary

The use of benzol in American industry has markedly decreased in the past six years and is still decreasing. Credit for this reform is largely due to the National Safety Council's investigation and the subsequent campaign of education among its members.

The greatest change is in the making of sanitary cans where benzol-rubber sealing compound is being rapidly replaced by rubber latex. Other occupations in which latex is displacing benzol-rubber are: making cord fabric for tires, cementing shoes, both rubber and leather, making dipped rubber and spread rubber goods.

Toluol is displacing benzol in nitrocellulose coatings and lacquers, while naphtha, or the less volatile petroleum distillate "Stoddard's Solvent," has largely displaced it in dry cleaning. Several state organizations of the painters' union have carried on suc-

cessful agitation against the use of benzol in quick-drying paints and other coatings.

Benzol is still generally used for coating patent leather, and sometimes artificial leather; for sealing bottles; for fastening rubber heels on leather soles; for tire building; and as a constituent of paint and varnish removers; but it is believed by insurance men and by many industrialists that in these processes also substitutes for benzol will eventually be found.

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LEAD POISONING FROM LEAD PIPED WATER SUPPLIES*

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MOST of the lead poisoning now recognized is attributed to the hazards of certain occupations. There are, however, many cases which cannot be traced to occupational exposure to lead but which are due rather to the use of water supplied through lead pipes. In the Industrial Clinic of the Massachusetts General Hospital it was found that almost 11 per cent. of the cases of lead poisoning studied over a period of many years (1911 to 1923) were of nonindustrial origin, for the most part associated with the consumption of lead piped waters, of home distilled alcohol, or of homemade wines. This group was, in size, second only to that of painters. In addition to the cases seen as hospital patients, many others were found upon local investigation of home conditions, a total number so impressive as to indicate that nonindustrial lead poisoning existed in New England as a public health problem of importance. As lead pipes are widely used in other parts of the country it was felt that a similar situation might be encountered elsewhere, particularly in regions supplying soft waters.

With the generous assistance of the Massachusetts Department of Public Health a brief study of the matter was carried on during the summer of 1923.[1] Visits were made to twenty-seven cities and towns of eastern and central Massachusetts (Table 1) and, with the co-operation of district health officers and of local physicians and plumbers, 102 sources of water supply were investigated.

In each home visited, information was secured regarding the water supply and the length of lead pipe used, and, in connection with ninety sources, clinical data were obtained concerning persons using each supply. The individual examinations included a search for a lead line, a hemoglobin determination by the Tallqvist scale, and the making of blood smears. Data regarding 253 persons were obtained and the findings recorded upon the form shown in Figure 1.

Among the adult males five or six were found whose occupations might

* Received for publication Feb. 21, 1928.

[1] Two of the authors, W. W. and C. O. S., held temporary appointments in the Department.

have offered some degree of exposure to lead but of these one only was poisoned. This man was a plumber who stated that he had not been occupationally exposed to lead for eight years. His wife was also poisoned.

It may be assumed that there was a fair random sampling of lead conducted water supplies as the efforts of the investigators were directed primarily toward the finding of lead piped sources of supply rather than toward the survey of conditions attending the development of known cases of lead

TABLE 1.—27 CITIES AND TOWNS IN MASSACHUSETTS IN WHICH LEAD CONDUCTED WATER SUPPLIES WERE STUDIED

CITY OR TOWN	WATER SOURCES STUDIED				WATER SOURCES IN CLINICAL INVESTIGATIONS		PERSONS EXAMINED	PERSONS POISONED
	Total	City	Well	Spring	Used	Poisonous		
Totals	102	72	20	10	90	35	253	63
Ashburnham	1	..	1	..	1	..	3	..
Bedford	6	..	6	..	6	3	15	3
Boxborough	1	..	1	..	1	1	1	1
Cambridge	1	1	..	..	1	1	5	3
Carlisle	1	..	1	..	..	..	..	..
Dana	2	..	..	2	2	2	5	3
Enfield	4	..	..	4	4	2	12	5
Greenwich	3	..	1	2	3	3	41	17
Hingham	4	4	..	..	4	1	9	1
Lexington	1	1	..	..	..	..	..	..
Lowell	13	13	..	..	11	5	36	9
Metropolitan District	(26)	(26)	..	..	(22)	(9)	(46)	(12)
Boston	6	6	..	..	3	2	9	3
Everett	1	1	..	..	1	1	5	1
Malden	6	6	..	..	6	2	12	3
Medford	1	1	..	..	1	..	2	..
Melrose	7	7	..	..	7	2	13	2
Quincy	4	4	..	..	3	1	3	1
Revere	1	1	..	..	1	1	2	2
Marlborough	6	6	..	..	5	..	10	..
Milford	6	6	..	..	5	3	5	3
Petersham	6	..	6	..	6	1	15	1
Reading	4	4	..	..	4	1	9	1
Royalston	1	..	1	..	1	1	5	1
Somerville	1	1	..	..	..	..	..	..
Wakefield	5	5	..	..	5	..	11	..
Warren	5	..	3	2	4	2	16	3
Wellesley	5	5	..	..	5	..	9	..

poisoning. Large cities, moderate-sized towns, and small villages were included. The economic status of those persons observed varied greatly.

Name.....Date seen.....Lead study #.....
For how long?.....
Address.....
Hist. of water supply.....
Medical attendant.....
Age.....M. F.....W. O.....S. M. W. D.....Nationality.....
Births.....Miscarriages.....Stillbirths.....Deaths.....
Pres. res.....
Industry.....Occupation.....
Dur. ind.....Dur. pres. trade.....Prev. emp.....
Complaint.....
Habits.....
Appetite...Constip....Diar....Naus....Vom....Eruct....Wt. loss...lbs. in.....
Abdominal pain-distress.....Where.....When.....
Memory.....Vision.....Headache.....Vertigo.....Ataxia.....Asthenia.....
Joint pain.....Paresthesia.....Strabismus.....
Pb line...Pallor...Weakness Flex....Ext....R. & L....Wr. Dr....Toe Dr.....
Blood Hgb.....RBC.....
Stippling.....Diff.....P.....LM.....LL.....SL.....M.....E.....Tr.....
Diagnosis.....

FIG. 1.—Record form.

CLINICAL INVESTIGATION

In the course of this house to house visitation, wholly unsolicited by the visited, it was not possible to make satisfactory and thorough clinical inquiries and examinations. It is certain that the recorded findings are incomplete. An astonishing number of persons, however, presented a definite picture of lead poisoning while in many others there were found such evidences suggestive of lead absorption or poisoning as to render it highly probable that careful and continued observation would have established the diagnosis.

The authors do not wish to assert dogmatically that unquestionable lead poisoning was found in a certain proportion of exposed persons but wish rather to present the facts as they

were observed, significant enough in themselves.

For purposes of tabulation and comparison there was selected a group of sixty-three cases which definitely or very probably were cases of poisoning, presenting, in addition to a lead line or stippling of the red blood cells, at least two symptoms common to cases of lead poisoning. It must be understood that in the absence of complete clinical records this selection is somewhat arbitrary, but it is believed that the factor of error is not a large one. By this method of selection many persons were excluded who presumably were poisoned but regarding whom sufficient data of record were not available. For example, twenty persons with stippled red blood cells and two with lead lines are not included among the poisoned. It is possible that more exhaustive study of the subjects would have resulted in additions to, or withdrawals from, the group of persons here identified as poisoned. It should also be remembered that, with few exceptions, all these cases of poisoning were very mild.

Of the sixty-three cases of poisoning, forty-two were related to the use of water supplies which each produced two or more cases of poisoning. Of the remaining twenty-one cases, eight were users of sources in connection with which but a single clinical observation was made. One source, a spring supplying a private school, produced eleven cases among twenty-four persons exposed; another spring, five cases among thirteen persons exposed.

Despite the fact that most of the cases of poisoning found were of a moderate or a slight degree of severity, they offered, in great majority, convincing clinical pictures. Several case histories are summarized in illustration.

CASE 11.—C. B., a housewife, 31 years of age, was born in the United States and had lived in Enfield for four years.

She showed a lead line and marked pallor, and gave a history of irregular distress low in the abdomen, of early pregnancy, and of occasional constipation.

Blood examination showed: hemoglobin 55 per cent.; stippling of the red cells; and normal leukocytes.

She used water from a spring containing 2.261 mg. of lead per liter. Her daily intake of lead was 6.215 mg.

Her husband and one child showed stippling.

CASE 142.—W. M., a schoolboy 15 years of age, was born in the United States and had lived in Lowell for six years.

He gave a history of slight indigestion and epigastric distress after meals, and of occasional headaches. Marked general weakness, enlargement of the finger joints, and contraction of the right little finger were noted.

Hemoglobin was 70 per cent. The red cells showed stippling and moderate variation in size and staining, with polychromatophilia.

He used water from the city supply which contained 0.485 mg. of lead per liter. His daily intake of lead was 1.81 mg.

Two younger sisters and a brother showed stippling.

CASE 175.—E. S., a housewife, aged 50 years, born in the United States, had lived in Lowell for forty-five years.

She complained until recently of weakness of the forearms. Her appetite was good. She gave a history of indigestion with eructations and epigastric distress, constipation, occasional headache, and vertigo. Marked asthenia, weakness of the flexors and extensors of both forearms, and lead line were noted.

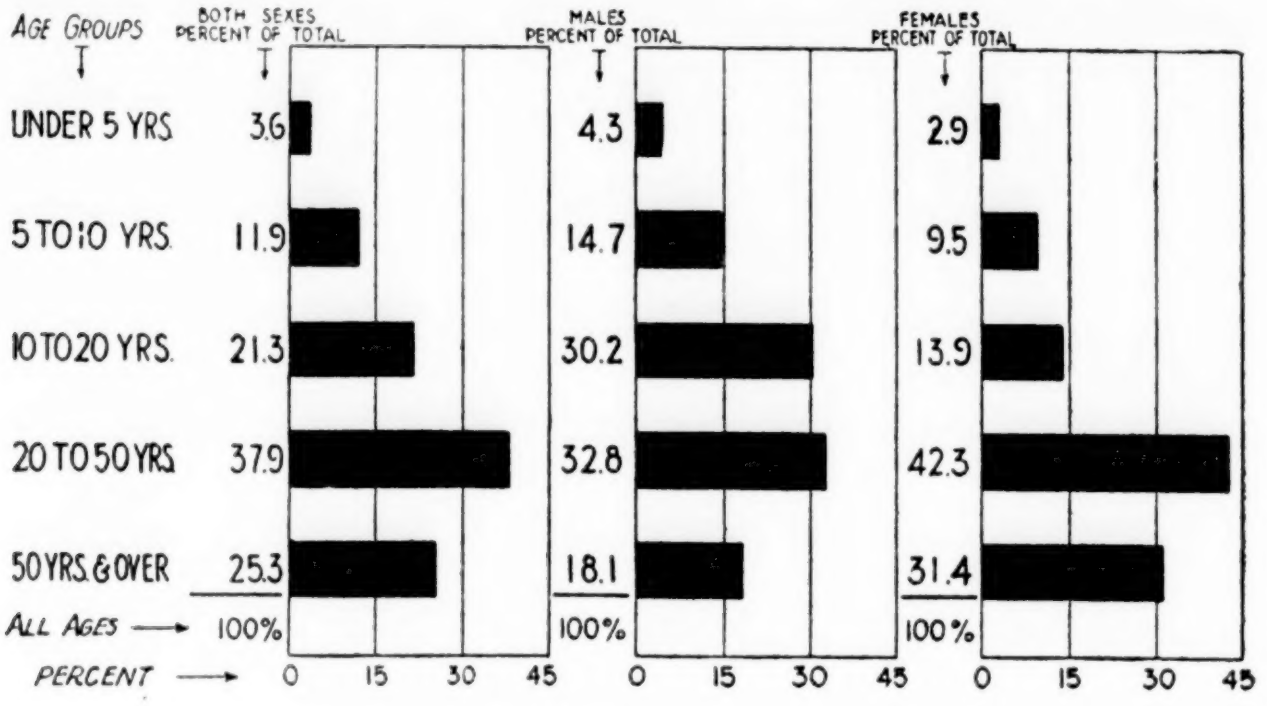
Blood examination showed: hemoglobin 60 per cent.; stippling of the red cells and marked variation in size and depth of staining; and polychromatophilia.

Until two years ago, she had used the city water supply, which came from Cooke wells.[2] Since then her source of supply has been a driven well from which the water runs through 20 feet of lead pipe. The lead content of this supply was 0.075 mg. per liter. The patient's daily intake of lead was 0.168 mg.

Her son, aged 14 years, showed stippling and other evidences of lead poisoning.

CASE 249.—T. K., a meat cutter, aged 29 years, born in Poland, had lived in Cambridge for five years.

He showed a marked lead line, marked pallor, and moderate weakness of the flexors of the wrists. He complained of epigastric hunger pain, constipation alternating with diarrhea, and headaches.



AGE GROUP	BOTH SEXES		MALES		FEMALES	
	No.	% Distribution	No.	% Distribution	No.	% Distribution
Totals	253	100.0	116	100.0	137	100.0
yrs.						
Under 5	9	3.6	5	4.3	4	2.9
5–10	30	11.9	17	14.7	13	9.5
10–20	54	21.3	35	30.2	19	13.9
20–50	96	37.9	38	32.8	58	42.3
50 and over	64	25.3	21	18.1	43	31.4

FIG. 2.—Distribution of exposed persons by sex and age groups.

Blood examination showed: hemoglobin 80 per cent.; stippled red cells; differential count: polymorphonuclears, 41 per cent.; large mononuclears, 11 per cent.; large lymphocytes, 27 per cent.; small lymphocytes, 19 per cent.; eosinophils, 1 per cent.; and transitionals, 1 per cent.

He used city water having a lead content of 0.0833 mg. per liter. His daily intake of lead was probably in excess of 0.05 mg.

His children also showed evidences of poisoning.

CASE 250.—R. L., an Italian, 38 years of age, had been a common laborer for twenty years. He had lived in Revere for three years.

He complained of poor appetite, persist-

[2] The city supply of Lowell occasionally included waters from wells. Water from the Cooke wells, at the source, contained 0.143 mg. of lead per liter.

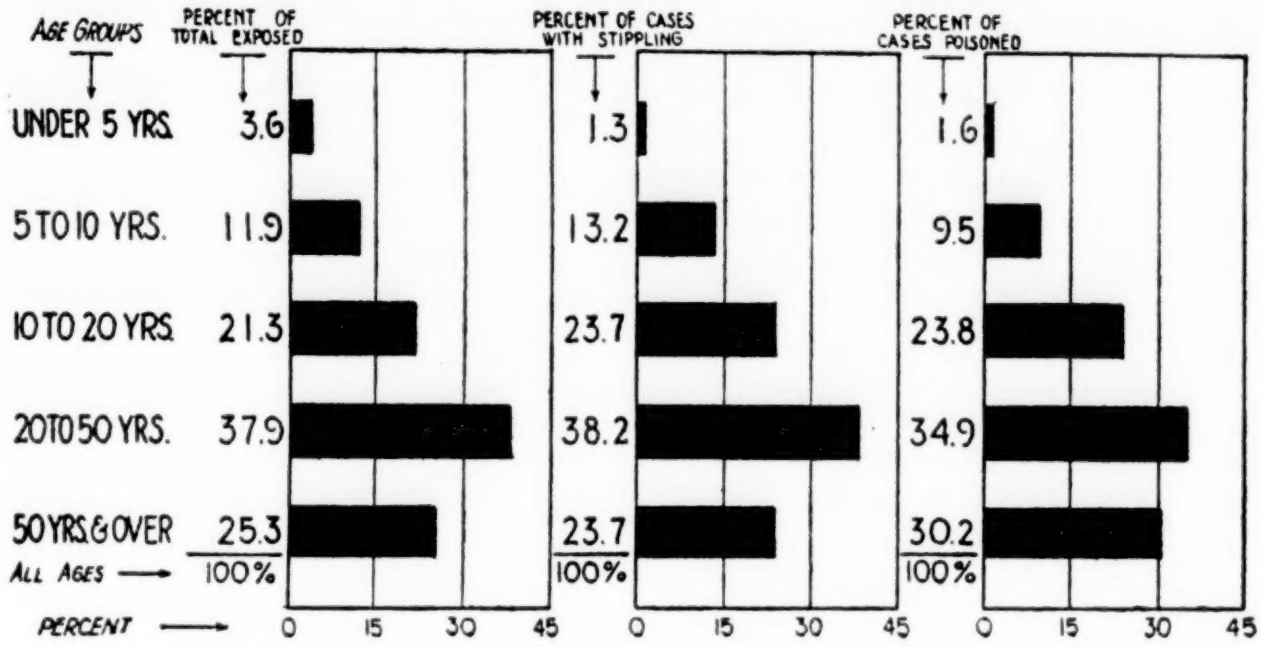
ent constipation, epigastric distress with occasional nausea and vomiting, and headache. A marked lead line, marked general weakness, and loss of power of both flexors and extensors of the forearms were noted. There was a question here of the possible use of alcohol.

Hemoglobin was 55 per cent. by the Dare scale, and 75 per cent. by the Tallqvist scale. Many stippled red cells were seen.

He used water from the city supply which contained 0.078 mg. of lead per liter. His daily intake of lead was probably in excess of 0.08 mg.

His wife had a lead line and gastro-intestinal manifestations of poisoning, and showed stippling of the red cells. She denied the use of wine.

Figure 2 presents the percentage distribution of the 253 exposed persons according to age groups, distinguishing sex. Though the selection of persons was largely determined by chance, observations were recorded with regard to approximately as many males as females and with regard to a fair proportion of children as compared with adults.



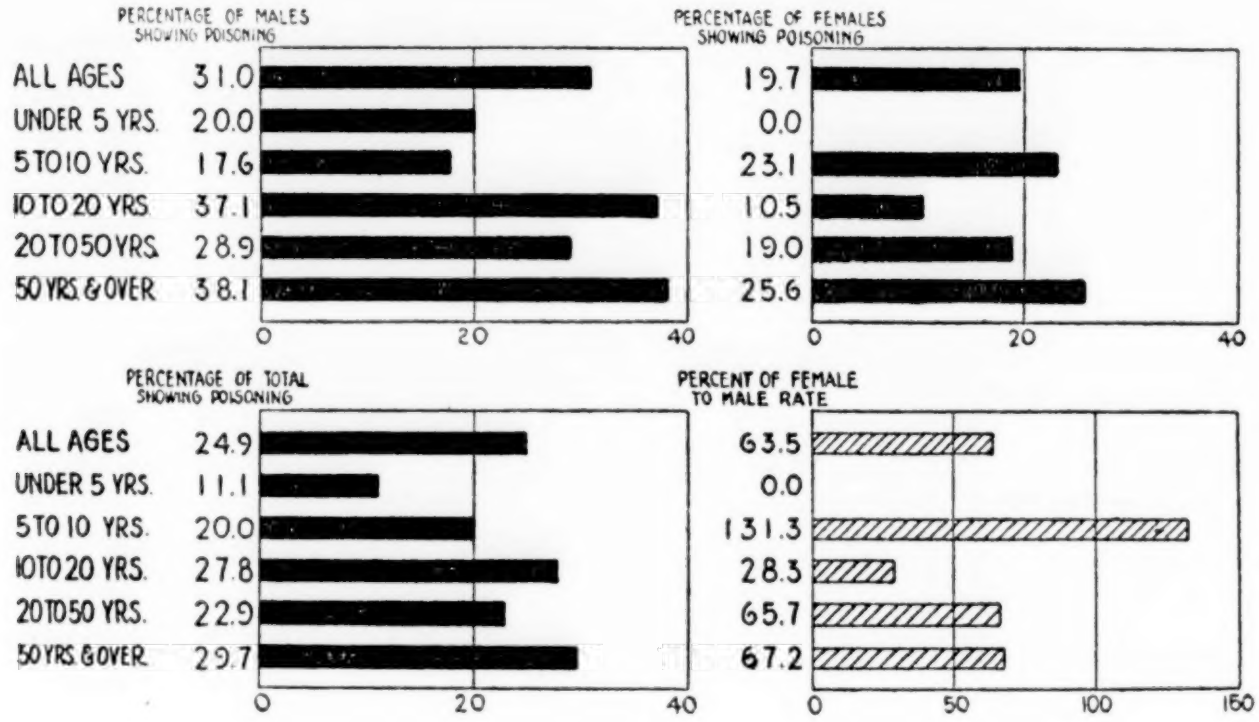
AGE GROUP	PERSONS EXPOSED		PERSONS SHOWING STIPPLING		PERSONS POISONED	
	No.	% Distribution	No.	% Distribution	No.	% Distribution
Totals	253	100.0	76	100.0	63	100.0
yrs.						
Under 5	9	3.6	1	1.3	1	1.6
5–10	30	11.9	10	13.2	6	9.5
10–20	54	21.3	18	23.7	15	23.8
20–50	96	37.9	29	38.2	22	34.9
50 and over	64	25.3	18	23.7	19	30.2

FIG. 3.—Distribution of exposed persons and of those poisoned, by age groups.

Figure 3 presents the percentage distribution by age groups of the 253 persons exposed, and of sixty-three cases of poisoning.

Figure 4 presents the percentage of those in each age group who show

poisoning, for each sex and for both sexes combined. It is noteworthy that



AGE GROUP	BOTH SEXES			MALES			FEMALES			% FEMALE OF MALE RATE OF POISONING
	No. Exposed	No. Poisoned	% Poisoned	No. Exposed	No. Poisoned	% Poisoned	No. Exposed	No. Poisoned	% Poisoned	
Totals	253	63	24.9	116	36	31.0	137	27	19.7	63.5
yrs.										
Under 5	9	1	11.1	5	1	20.0	4	0	0.0	0.0
5–10	30	6	20.0	17	3	17.6	13	3	23.1	131.3
10–20	54	15	27.8	35	13	37.1	19	2	10.5	28.3
20–50	96	22	22.9	38	11	28.9	58	11	19.0	65.7
50 and over	64	19	29.7	21	8	38.1	43	11	25.6	67.2

FIG. 4.—Percentage of poisoning in each age group, by sex.

except in one age group, 5 to 10 years, females showed a distinctly lower incidence of poisoning than did males. It will be found later that a similar relationship generally prevails when the two sexes are compared.

In Table 2 are listed the principal symptoms and signs recorded, setting forth the number of times each was encountered, the percentage incidence, the number of times each appeared in the group of sixty-three cases of poisoning, and the percentage incidence in that group. The incidence of eructations is tabulated not because the symptom is notably pathognomonic of lead poisoning, but because of its importance as evidence of digestive disturbance and of the certainty with which a history of the symptom and its severity can be obtained.

Table 3 presents the percentage distribution of certain symptoms by age groups and also the incidence in the respective age groups. The apparently low incidence of various

symptoms among exposed persons of the two lower age groups may be due to the difficulty experienced in obtaining satisfactory histories from children, or possibly, among children of the first age group, to a diet rich in milk. The effect of a high calcium diet in binding lead in the bones has been ably demonstrated by Aub.

TABLE 2.—PRINCIPAL SYMPTOMS AND SIGNS FOUND AMONG 253 PERSONS EXAMINED

FINDING	TOTAL CASES		CASES WITH POISONING	
	No.	%	No.	%
Totals	253	100.0	63	100.0
Pallor	157	62.1	47	74.6
Hemoglobin less than 70%	82	32.4	25	39.7
Constipation	78	30.8	25	39.7
Eructations	77	30.4	26	41.3
Stippling	76	30.0	56	88.9
Headache	73	28.9	33	52.4
Joint pain	57	22.5	14	22.2
Abdominal pain	53	20.9	22	34.9
Vertigo	36	14.2	9	14.3
Lead line	26	10.3	24	38.1
Weakness in forearm	26	10.3	14	22.2
Loss of appetite	21	8.3	9	14.2
Loss of weight	15	5.9	8	12.7

A lead line is relatively rarely found in the mouth of a child unless the condition of the teeth and gums is unusually poor. Stippling, also a wholly objective finding, was quite uniformly high except in the first age group.

Data were obtained regarding the approximate time each person exposed had used the water supply under consideration. Figure 5 presents the percentage distribution of the entire group of exposed persons according to periods of exposure, and the incidence of poisoning in each subgroup. The duration of exposure does not seem to be a factor of great importance in the production of poisoning, as a greater incidence was found among those exposed from three to five years than among those exposed over ten years.

Figure 6 shows a similar distribution for the sixty-three cases of poisoning and also separately for adults, considered as those persons aged 20 and more, and for children under 20 years of age. There is no evidence of excessive susceptibility of children.

Table 4 in more detail presents, according to periods of exposure, the incidence of poisoning for each exposure group and the findings among children and male and female adults. Children under 10 years of age showed a lower incidence of poisoning than did those from 10 to 20 years old; children 10 to 20 a lower incidence than adult males, but a higher incidence than did adult females or all adults combined. The adult female rate for all periods of exposure was 67.7 per cent. of the male rate. For one period only was it higher than the male rate. Female children from 10 to 20 years of age have not been included among adult females despite the fact that many of them were to a great degree functionally mature. Girls of this age group showed 10.5 per cent. poisoned as against 37.4 per cent. among boys, or 28 per cent. of the male rate. If grouped with older females, girls would force still lower the relatively low incidence of poisoning among women as compared with men.

There was no evidence brought forth in this study to support the tra-

TABLE 3.—NUMBER AND PERCENTAGE OF CASES, AND PERCENTAGE DISTRIBUTION, BY AGE GROUPS, OF CERTAIN CLINICAL FINDINGS AMONG 253 PERSONS EXPOSED TO LEAD IN WATER

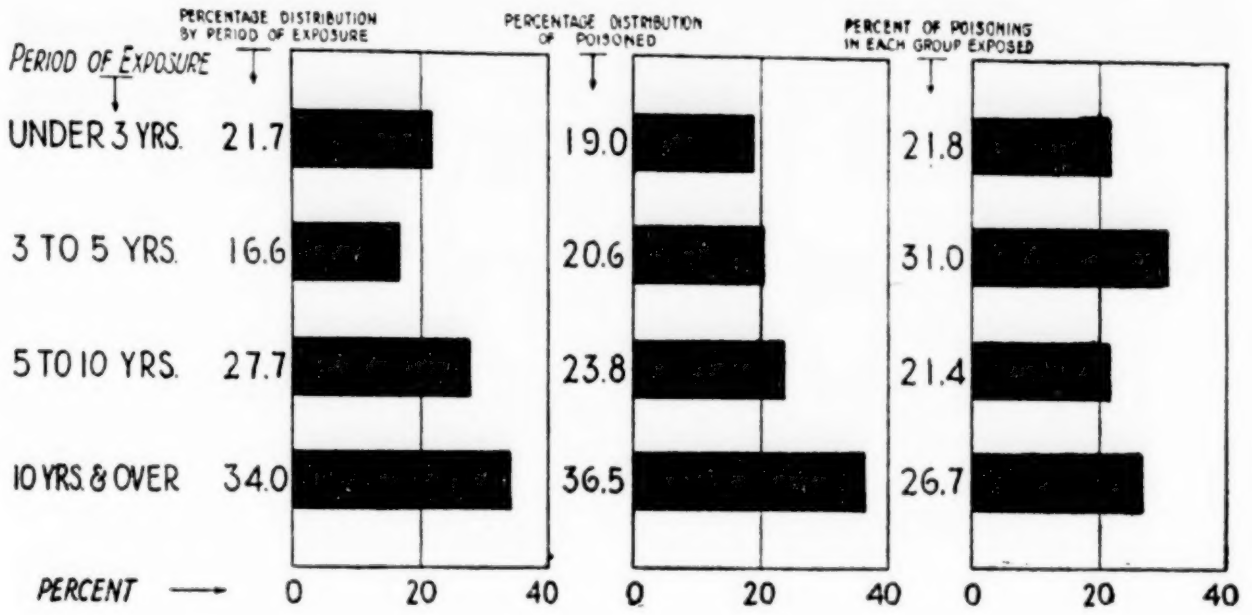
AGE GROUP	PERSONS EXPOSED	CASES WITH HEMOGLOBIN LESS THAN 70 %			CASES WITH STIPPLING			CASES WITH LEAD LINE			CASES WITH ERUCTATIONS			CASES WITH ABDOMINAL PAIN			CASES WITH CONSTIPATION		
		No.	% Distribution	% of Exposed	No.	% Distribution	% of Exposed	No.	% Distribution	% of Exposed	No.	% Distribution	% of Exposed	No.	% Distribution	% of Exposed	No.	% Distribution	% of Exposed
Totals.....	253	82	100.0	32.4	76	100.0	30.0	26	100.0	10.3	77	100.0	30.4	53	100.0	20.9	78	100.0	30.8
yrs.																			
Under 5.....	9	6	7.3	66.7	1	1.3	11.1	0	0.0	0.0	0	0.0	0.0	0	0.0	0.0	3	3.8	33.3
5–10.....	30	10	12.1	33.3	10	13.2	33.3	1	3.8	3.3	1	1.3	3.3	5	9.4	16.7	1	1.3	3.3
10–20.....	54	24	29.3	44.4	18	23.7	33.3	1	3.8	1.9	15	19.5	27.8	11	20.8	20.4	10	12.8	18.5
20–50.....	96	23	28.0	24.0	29	38.2	30.2	16	61.5	16.7	29	37.7	30.2	23	43.3	24.0	37	47.4	38.6
50 and over....	64	19	23.2	29.7	18	23.7	28.1	8	30.8	12.5	32	41.6	50.0	14	26.4	21.9	27	34.6	42.2

TABLE 4.—INCIDENCE OF POISONING AMONG CHILDREN AND MALE AND FEMALE ADULTS WITH RELATION TO DURATION OF EXPOSURE

PERIOD OF EXPOSURE	CHILDREN UNDER 10 YRS.			CHILDREN 10–20 YRS.			ADULTS OF BOTH SEXES			MALE ADULTS			FEMALE ADULTS			% ADULT FEMALE OF ADULT MALE RATE OF POISONING
	No. Exposed	No. Poisoned	% Poisoned	No. Exposed	No. Poisoned	% Poisoned	No. Exposed	No. Poisoned	% Poisoned	No. Exposed	No. Poisoned	% Poisoned	No. Exposed	No. Poisoned	% Poisoned	
Totals.....	39	7	17.9	54	15	27.8	160	41	25.6	59	19	32.2	101	22	21.8	67.7
yrs.																
Under 3.....	13	2	15.3	13	4	30.8	29	6	20.7	11	3	27.3	18	3	16.7	61.2
3–5.....	7	1	14.3	11	4	36.4	24	8	33.3	10	2	20.0	14	6	42.9	214.5
5–10.....	19	4	21.1	16	4	25.0	35	7	20.0	14	7	50.0	21	0[1]	0.0	0.0
10 and over....	0	0	0.0	14	3	21.4	72	20	27.8	24	7	29.2	48	13	27.1	92.8

[1] Eight of this group were clearly borderline cases.

ditional and, perhaps, apocryphal belief that women are more susceptible to lead poisoning than men or that because of exposure to lead they are subject to miscarriages or sterility. Sixty-three married women were exposed to lead during the child bearing period; fifteen were lead poisoned. Of the sixty-three, thirty-two bore fifty-five children, with but one miscarriage. Of the thirty-two mothers, eight who were poisoned bore thirteen children. Thirty-one women, seven of them poisoned, bore no children during the period of exposure to lead but of the thirty-one, ten were previously barren and of the remaining twenty-one fertile women, four of whom were poisoned, many were near the menopause or already possessed of many children or so recently mothers or had been exposed to lead for so short a period that no significance can be attached to the association of childlessness and lead ingestion.



PERIOD OF EXPOSURE	PERSONS EXPOSED		PERSONS POISONED		
	No.	% Distribution	No.	% Distribution	% of Exposed
Totals	253	100.0	63	100.0	24.9
yrs.					
Under 3	55	21.7	12	19.0	21.8
3–5	42	16.6	13	20.6	31.0
5–10	70	27.7	15	23.8	21.4
10 and over	86	34.0	23	36.5	26.7

FIG. 5.—Distribution of exposed persons and of those poisoned, by periods of exposure, and incidence of poisoning in each class.

Figure 7 presents the percentage distribution of the 253 persons exposed and of the sixty-three poisoned, according to the amount of lead ingested daily, assuming that the average daily fluid intake was that recorded for each person, and that the lead content of the water was uniform. It also shows the percentage of each subgroup found poisoned. There was no notable variation in the incidence of poisoning

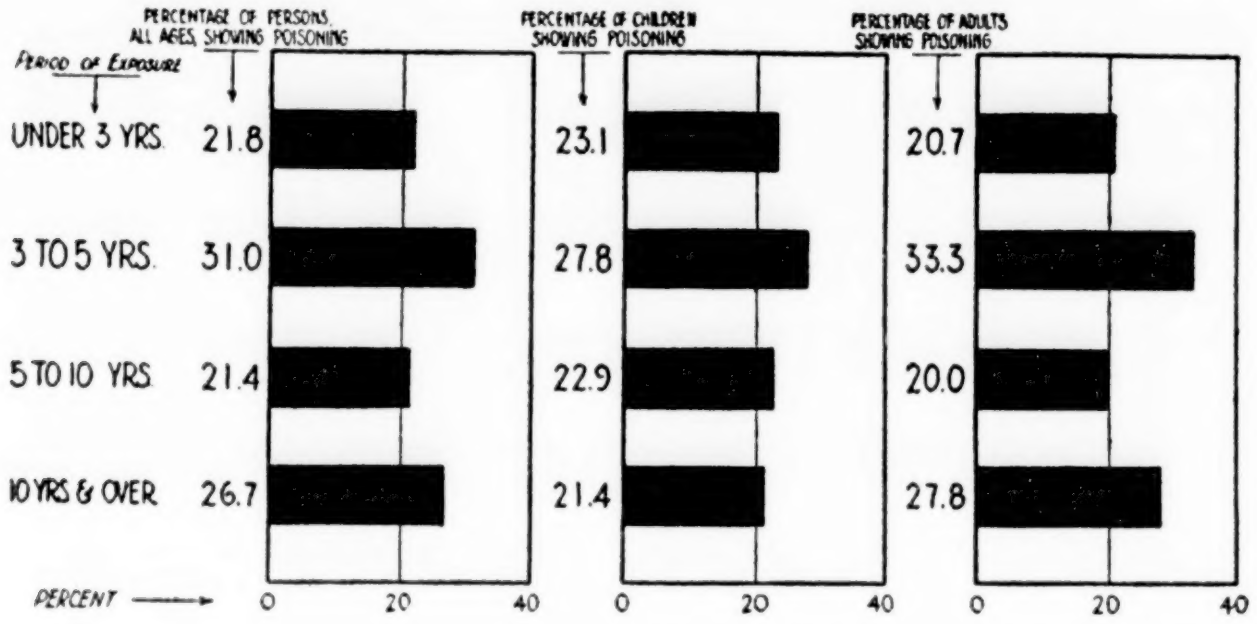
* Eight of this group were clearly borderline cases.

produced among those exposed to daily amounts of from less than 0.1 mg. to 1.5 mg. of lead, an incidence ranging from 20.5 to 25 per cent.; but of those ingesting daily over 1.5 mg., 50 per cent. showed poisoning.

In Table 5 is shown the incidence of poisoning found associated with the daily ingestion of various amounts, for children and for adults of each sex. Here again the adult female incidence of poisoning for all degrees of dosage but one is lower than the adult male rate.

Figure 8 presents the distribution of the 253 persons exposed, by estimated total lead intake over the entire period of exposure to lead; the distribution of the sixty-three cases with poisoning, by total lead intake; and the percentage of each subgroup of exposed persons who showed poisoning. There is quite close agreement between the percentage distribution of the exposures and of the cases poisoned, suggesting simply that where there were many exposed there were, reasonably enough, many poisoned. There was, however, an increasing liability to poisoning as the total amount ingested increased.

Table 5 presents the incidence of poisoning in each subgroup (classified by total amounts ingested) among



PERIOD OF EXPOSURE	ALL AGES			CHILDREN UNDER 20 YRS.			ADULTS		
	No. Exposed	No. Poisoned	% Poisoned	No. Exposed	No. Poisoned	% Poisoned	No. Exposed	No. Poisoned	% Poisoned
Totals	253	63	24.9	93	22	23.7	160	41	25.6
yrs.									
Under 3	55	12	21.8	26	6	23.1	29	6	20.7
3–5	42	13	31.0	18	5	27.8	24	8	33.3
5–10	70	15	21.4	35	8	22.9	35	7	20.0
10 and over	86	23	26.7	14	3	21.4	72	20	27.8

FIG. 6.—Incidence of poisoning among exposed persons, by periods of exposure.

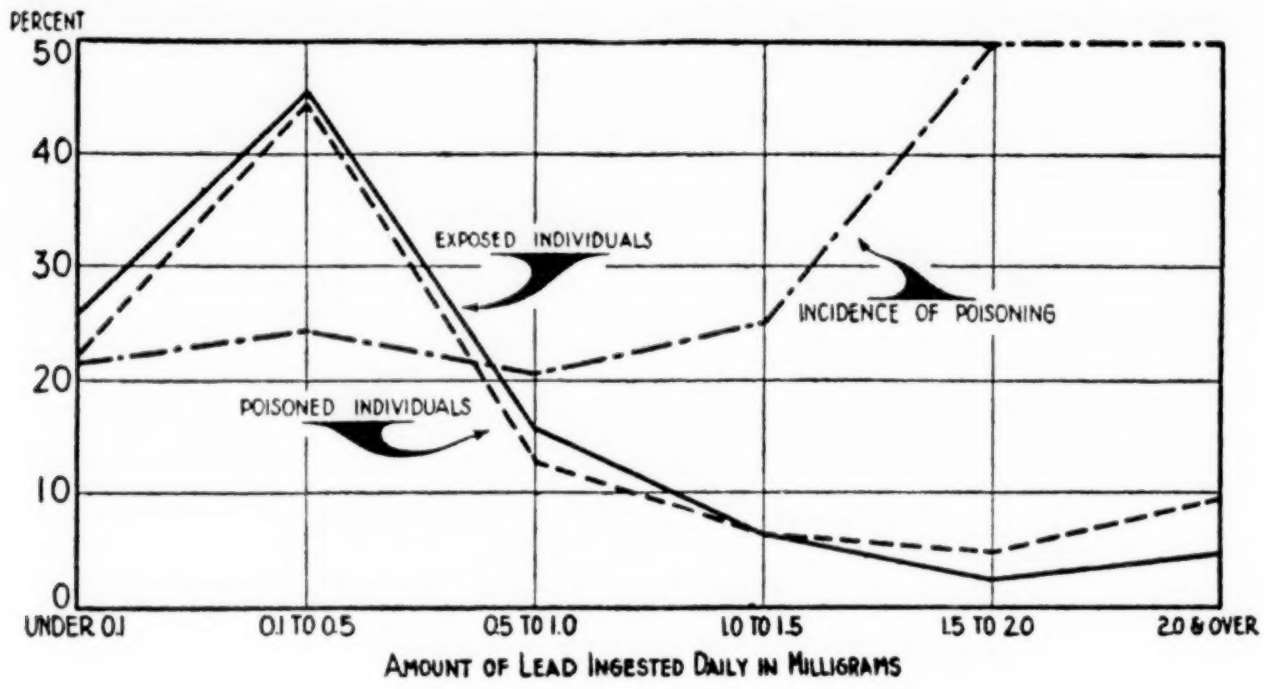
TABLE 5.—DISTRIBUTION OF 253 CASES BY AMOUNTS OF LEAD INGESTED, FURTHER CLASSIFIED AS CHILDREN, MALE ADULTS, AND FEMALE ADULTS, WITH INCIDENCE OF POISONING

AMOUNT OF LEAD INGESTED *mg.*	ALL AGES		CHILDREN UNDER 20 YRS.					ADULT MALES					ADULT FEMALES					% ADULT FEMALE OF ADULT MALE RATE OF POISONING
			Exposed		Poisoned			Exposed		Poisoned			Exposed		Poisoned			
	No. Exposed	% Distribution	No.	% Distribution	No.	% Distribution	% of Exposed	No.	% Distribution	No.	% Distribution	% of Exposed	No.	% Distribution	No.	% Distribution	% of Exposed	
Totals	253	100.0	93	100.0	22	100.0	23.7	59	100.0	19	100.0	32.2	101	100.0	22	100.0	21.8	67.7
Daily																		
Under 0.1	65	25.7	27	29.0	5	22.7	18.5	16	27.1	6	31.6	37.5	22	21.8	3	13.6	13.6	36.3
0.1–0.5	115	45.5	43	46.2	9	40.9	20.9	25	42.4	8	42.1	32.0	47	46.5	11	50.0	23.4	73.1
0.5–1	39	15.4	12	12.9	6	27.3	50.0	10	16.9	1	5.3	10.0	17	16.8	1	4.5	5.9	59.0
1–1.5	16	6.3	7	7.5	0	0.0	0.0	2	3.4	1	5.3	50.0	7	6.9	3	13.6	42.9	85.8
1.5–2	6	2.4	2	2.2	1	4.5	50.0	1	1.7	1	5.3	100.0	3	3.0	1	4.5	33.3	33.3
2 and over	12	4.7	2	2.2	1	4.5	50.0	5	8.5	2	10.5	40.0	5	5.0	3	13.6	60.0	150.0
Total																		
Under 500	133	52.6	63	67.7	14	63.6	22.2	25	42.4	7	36.8	28.0	45	44.6	8	36.4	17.8	63.7
500–1,000	34	13.4	10	10.7	2	9.1	20.0	11	18.6	3	15.8	27.3	13	12.9	1	4.5	7.7	28.2
1,000–2,000	35	13.8	8	8.6	3	13.6	37.5	9	15.3	4	21.1	44.4	18	17.8	4	18.2	22.2	50.0
2,000–5,000	29	11.5	10	10.7	2	9.1	20.0	7	11.9	2	10.5	28.6	12	11.9	3	13.6	25.0	87.4
5,000–10,000	9	3.6	1	1.1	1	4.5	100.0	1	1.7	1	5.3	100.0	7	6.9	2	9.1	28.6	28.6
10,000 and over	13	5.1	1[1]	1.1	0	0.0	0.0	6	10.2	2	10.5	33.3	6	5.9	4	18.2	66.7	200.3

[1] A girl 19 years of age, with numerous symptoms attributable to lead poisoning but excluded under the conditions previously stipulated.

children and among adult males and females. As noted before, the male rate is in excess of the female rate except for one small classification.

consumed. It also gives the percentage of each group of exposed persons showing poisoning. It may be noted that about 88 per cent. of the sources, which were responsible for 84 per cent. of the cases of poisoning, furnished water with a lead content of less than 0.5 mg. per liter. The case incidence among exposed persons, however, was highest among those using water containing over 1 mg. per liter.

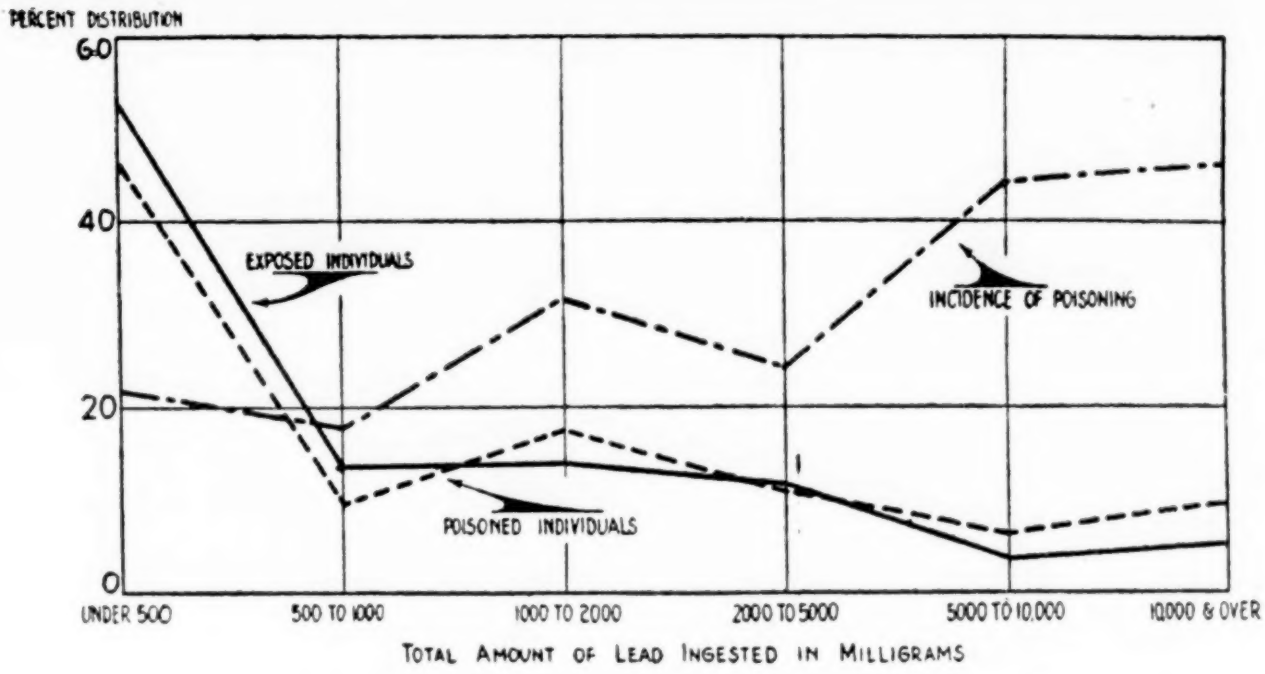


AMOUNT OF LEAD INGESTED DAILY	PERSONS EXPOSED		PERSONS POISONED		
	No.	% Distribution	No.	% Distribution	% of Exposed
Totals	253	100.0	63	100.0	24.9
mg.					
Under 0.1	65	25.7	14	22.2	21.5
0.1–0.5	115	45.5	28	44.4	24.3
0.5–1	39	15.4	8	12.7	20.5
1–1.5	16	6.3	4	6.3	25.0
1.5–2	6	2.4	3	4.8	50.0
2 and over	12	4.7	6	9.5	50.0

FIG. 7.—Distribution of exposed persons and of those poisoned, by amounts of lead ingested daily, and incidence of poisoning in each class.

Figure 9 presents the percentage distribution of ninety sources of water supply according to their lead contents, expressed in milligrams per liter; of the 253 persons exposed, according to the lead content of the waters consumed; of the thirty-five sources which were productive of poisoning, according to their lead contents; and of the sixty-three persons poisoned, according to the lead content of waters

Table 6 presents the percentage distribution of ninety sources according to the lead content in milligrams per liter, divided as city or town



TOTAL AMOUNT OF LEAD INGESTED	PERSONS EXPOSED		PERSONS POISONED		
	No.	% Distribution	No.	% Distribution	% of Exposed
Totals	253	100.0	63	100.0	24.9
mg.					
Under 500	133	52.6	29	46.0	21.8
500–1,000	34	13.4	6	9.5	17.6
1,000–2,000	35	13.8	11	17.5	31.4
2,000–5,000	29	11.5	7	11.1	24.1
5,000–10,000	9	3.6	4	6.3	44.4
10,000 and over	13	5.1	6	9.5	46.2

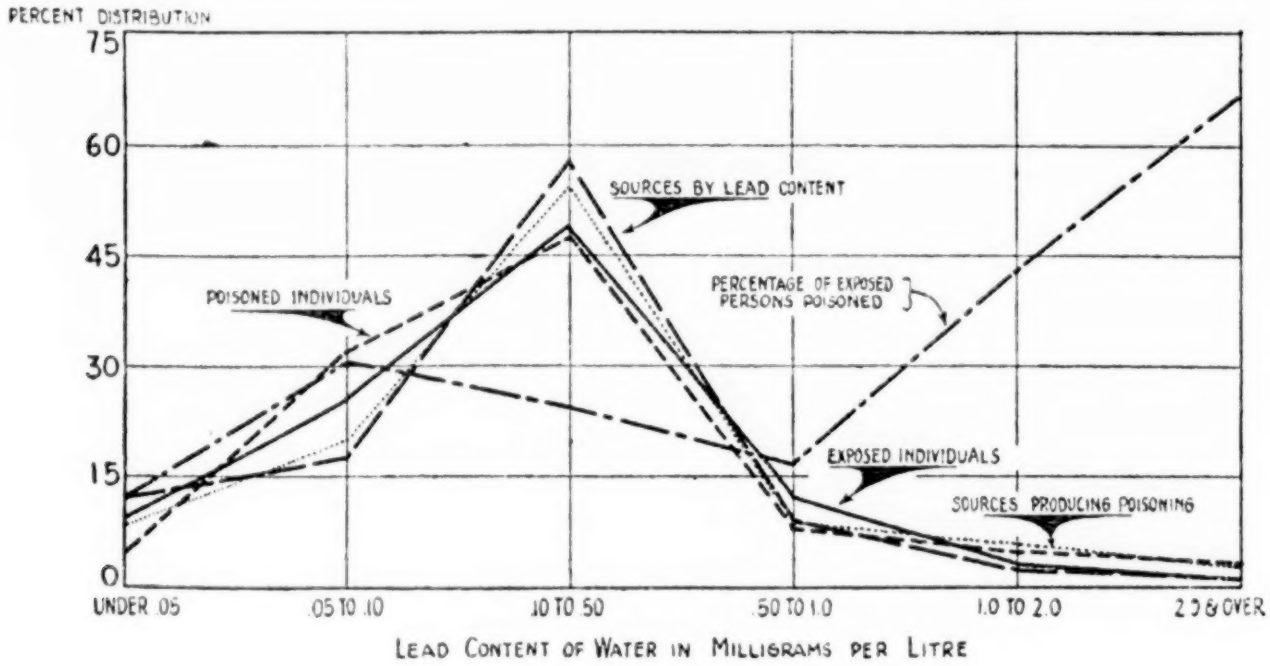
FIG. 8.—Distribution of exposed persons and of those poisoned, by total lead ingested, and incidence of poisoning in each class.

TABLE 6.—DISTRIBUTION OF WATERS OF VARYING DEGREES OF LEAD CONTENT AS CITY SUPPLIES, WELL WATERS, AND SPRING WATERS

LEAD CONTENT OF WATER	ALL SOURCES		CITY SUPPLIES			WELL WATERS			SPRING WATERS		
	No.	% Distribution	No.	% Distribution	% of All Sources	No.	% Distribution	% of All Sources	No.	% Distribution	% of All Sources
Totals	90	100.0	62	100.0	68.9	18	100.0	20.0	10	100.0	11.1
mg./l.											
Under 0.05	11	12.2	9	14.5	81.8	2	11.1	18.2	0	0.0	0.0
0.05 to 0.1	16	17.8	13	21.0	81.3	2	11.1	12.5	1	10.0	6.3
0.1 to 0.5	52	57.8	33	53.2	63.5	14	77.8	26.9	5	50.0	9.6
0.5 to 1	8	8.9	6	9.7	75.0	0	0.0	0.0	2	20.0	25.0
1 to 2	2	2.2	1	1.6	50.0	0	0.0	0.0	1	10.0	50.0
2 and over	1	1.1	0	0.0	0.0	0	0.0	0.0	1	10.0	100.0

supplies, wells, and springs. About 69 per cent. of the sources were city or town supplies; 20 per cent., wells; and 11 per cent., springs.[3] Eighty-nine per cent. of the city supplies and all the well water contained less than 0.5 mg. of lead per liter; 90 per cent. of the spring waters contained over 0.1 mg.

In Tables 7 and 8 are presented data relating to the sources of the various waters studied, with special consideration of those productive of poisoning. Table 7, which indicates the percentage of waters of each type of source which produced poisoning, shows clearly that the spring waters were more hazardous than the well waters, and these in turn more dangerous than the city and town supplies. It is further true, as shown in Table 8, that the incidence of lead poisoning among users of spring waters was about 223



LEAD CONTENT OF WATER	ALL SOURCES		PERSONS EXPOSED		SOURCES PRODUCING POISONING		PERSONS POISONED		
	No.	% Distribution	No.	% Distribution	No.	% Distribution	No.	% Distribution	% of Exposed
Totals	90	100.0	253	100.0	35	100.0	63	100.0	24.9
mg./l.									
Under 0.05	11	12.2	24	9.5	3	8.6	3	4.8	12.5
0.05–0.1	16	17.8	65	25.7	7	20.0	20	31.7	30.8
0.1–0.5	52	57.8	124	49.0	19	54.3	30	47.6	24.2
0.5–1	8	8.9	30	11.9	3	8.6	5	7.9	16.7
1–2	2	2.2	7	2.8	2	5.7	3	4.8	42.9
2 and over	1	1.1	3	1.2	1	2.9	2	3.2	66.7

FIG. 9.—Distribution of sources, of exposed persons, of sources producing poisoning, and of persons poisoned, by lead content of water.

[3] Part of the water supply of Lowell is from wells and the samples obtained in Lowell showed the characteristic high carbon dioxide content of ground water.

per cent. of that among consumers of well waters, and 187 per cent. of that among persons using city supplies.

TABLE 7.—DISTRIBUTION OF ALL SOURCES AND OF SOURCES PRODUCING POISONING AS CITY SUPPLIES, WELLS, OR SPRINGS, AND PERCENTAGE OF EACH TYPE PRODUCING POISONING

WATER SUPPLY	ALL SOURCES		SOURCES PRODUCING POISONING		
	No.	% Distribution	No.	% Distribution	% of All Sources
Totals	90	100.0	35	100.0	38.9
City	62	68.9	20	57.1	32.2
Well	18	20.0	8	22.9	44.4
Spring	10	11.1	7	20.0	70.0

TABLE 8.—DISTRIBUTION OF 253 PERSONS USING WATERS OF THE SEVERAL TYPES OF SOURCES, AND OF THE PERSONS THUS POISONED, AND THE INCIDENCE OF POISONING BY TYPE OF SOURCE

WATER SUPPLY	PERSONS EXPOSED		PERSONS POISONED		
	No.	% Distribution	No.	% Distribution	% of Exposed
Totals	253	100.0	63	100.0	24.9
City	140	55.3	29	46.0	20.7
Well	46	18.2	8	12.7	17.4
Spring	67	26.5	26	41.3	38.8

Blood Studies

Supplementing the clinical observations, two blood smears were made in each case. One was stained with

TABLE 9.—AVERAGES OF DIFFERENTIAL BLOOD COUNTS

GROUP	POLYMORPHONUCLEARS	LARGE MONONUCLEARS	LARGE LYMPHOCYTES	SMALL LYMPHOCYTES	MAST CELLS	EOSINOPHILS	TRANSITIONALS
	%	%	%	%	%	%	%
253 cases exposed	53.3	2.4	8.6	30.6	0.5	2.6	1.7
63 cases of poisoning	54.3	2.5	8.2	29.8	0.7	2.9	1.5

TABLE 10.—INCIDENCE OF LOW POLYMORPHONUCLEAR LEUKOCYTE COUNTS AND OF LYMPHOCYTOSIS

% OF WHITE CELL COUNT	CASES AMONG 253 PERSONS EXPOSED		CASES AMONG 63 PERSONS POISONED	
	No.	%	No.	%
Polymorphonuclear Leukocytes				
Less than 50	86	34.0	22	34.9
Less than 45	54	21.3	13	20.6
Less than 40	24	9.5	4	6.3
Small Lymphocytes				
40 and over	57	22.5	11	17.5
50 and over	15	5.9	4	6.3
Large and Small Lymphocytes				
50 and over	50	19.8	12	19.0
60 and over	8	3.2	2	3.2

Wright's stain, the other with Unna's polychrome methylene blue.

Stippling of red blood cells was found in the blood of seventy-six persons, and was usually associated with more or less marked polychromatophilia, and with variation in the depth of staining and in the size of the red cells.

Differential leukocyte counts showed, broadly speaking, a somewhat abnormal tendency toward a low polymorphonuclear leukocyte count and a high lymphocytosis. Data were not obtained regarding actual numbers of leukocytes and so it cannot be said that the lymphocytosis was relative or absolute.

The incidence of low polymorphonuclear leukocyte counts or of high lymphocytosis bore no evident relation to the presence of stippling or of signs of poisoning. It seemed to be independent of periods of exposure or amounts of lead ingested. It was associated with low hemoglobin estimations and was apparently related to sex, such abnormalities of the blood being more frequently observed in women and children than in men.

The averages of the differential counts are given in Table 9. Table 10 presents the findings concerning the incidence of low polymorphonuclear leukocyte (differential) counts and of lymphocytosis for the entire group of 253 persons exposed and for the sixty-three cases of poisoning.

The blood studies, apart from the findings relating to red cells, are quite inconclusive.

Chemical Investigations

For many years, centuries indeed, the effect of various waters upon lead pipes has been the subject of extended investigation. Even a very superficial outline of the studies conducted in this country and elsewhere could not but be too lengthy for this occasion. In order, however, that the current view regarding the nature of corrosion of water conducting pipes may be stated, the following quotation is presented from a recent paper by H. W. Clark, chief chemist of the Massachusetts Department of Health:

> The molecule of water is considered as an active substance with the power of dissociation; that is, pure water contains in addition to the molecule H_2O a number of hydrogen ions and hydroxyl ions, the hydrogen ions being positively charged, the hydroxyl ions negatively and hence an electric balance or equilibrium results. When a metal is immersed in water it discharges into this water a certain number of minute particles positively charged, that is, metal ions. These positively charged metal ions destroy the electrical equilibrium of the water and positively charged hydrogen ions pass to the metal, and there lose their positive charge and form a coating of hydrogen gas over the metal protecting it from further action unless oxygen is present in the water. If this is so water is formed by chemical reaction between the hydrogen film and the oxygen. The metal is again exposed, metal ions go into solution and the process of corrosion continues. This continues as long as there are hydrogen ions to neutralize the metal and enough free oxygen to remove the hydrogen film.
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> The part of carbonic acid in this modern theory comes in in this way. CO_2 forms a weak acid in water which tends to increase the hydrogen ions of the water. Some of this carbonic acid may be needed to hold in solution carbonates of calcium and magnesium but the remainder is free carbonic acid or in the modern language aggressive acid owing to the hydrogen ion increase. Hard waters in which the content of bicarbonates is large can contain fairly high amounts of carbon dioxide without any of it becoming aggressive as it is employed in

holding these bicarbonates in solution but soft waters free or comparatively free from these bicarbonates become aggressive even when containing relatively small quantities of the acid.

Clark has long regarded carbon dioxide in water as the principal factor in corrosion. It will later be seen that the results of the present study confirm this belief.

The carbon dioxide and oxygen contents and the alkalinity of each water were determined at the tap by a portable apparatus designed by Fred B. Forbes of the Massachusetts Department of Public Health and lent by that Department. Gallon samples of "running water" were obtained for the quantitative estimation of lead, and in many instances an additional sample which had stood overnight in lead pipes. The lead content of running and standing samples were averaged for the purposes of this paper.

The analysis of each of the waters for lead was carried out by one of two methods. The standard method of the American Public Health Association was used by the State Water Laboratory, and Fairhall's method by one of the authors (C. O. S.) after comparison had shown no notable difference in results where little organic matter was present. Both methods were occasionally employed on similar samples. In order to eliminate chance of error in the determinations, many of the residues were subjected to the microchemical test of Behrens and Kley as modified by Fairhall, which is efficient in identifying lead when present even in very minute quantities.

The greatest amount of lead found in any sample among those drawn from the 102 sources studied was 4.321 mg. per liter in a spring water; the smallest amount, 0.01 mg. in a well water.

The well waters and spring waters had the same average carbon dioxide content of 2.58 parts per 100,000; the city or town supplies, 0.74 parts per 100,000. If there be excluded from the city supplies the thirteen Lowell samples, largely well waters averaging 1.56 parts per 100,000, the carbon dioxide content of the remaining fifty-nine samples averaged 0.56 parts per 100,000.

The dissolved oxygen varied greatly even among samples from a single type of source. Similarly the alkalinity varied greatly among the waters of each class, though tending toward a lower level among city waters than among well or spring waters. It should be kept in mind that these observations relate to waters which have passed through lead pipe to the tap.

Because of the limited number of water samples available for study, it is evident that fairly high dependencies must be found if the several relations of the findings, one to another, are to be considered statistically significant.

Association coefficients were computed for the relations of the several groups of variables: lead content of water, length of pipe, carbon dioxide content, dissolved oxygen, and alkalinity, as determined in connection with 100 samples. Many of the relationships were apparently without significance, notably the relation of length of lead pipe to lead content of

water; of lead content to oxygen content; and of lead content to alkalinity. The positive associations were between the following:

1. High lead content and high carbon dioxide content, 0.64 ± 0.05.
2. High lead content, long pipe, and high carbon dioxide content, 0.71 ± 0.06.
3. High lead content and high carbon dioxide content, short pipe held constant, 0.59 ± 0.02.
4. Long pipe and low alkalinity, 0.66 ± 0.05.

Summary

A chemical study was made of 102 lead conducted water supplies—city, well, and spring waters—ninety of which were used by a total of 253 persons subjected to clinical investigation.

All the waters analyzed contained lead.

The lead content was most strikingly related to the carbon dioxide content.

There was no apparent relation between length of pipe and lead content.

Of ninety sources used by the persons studied, thirty-five caused poisoning as determined by certain criteria.

Of 253 exposed persons, sixty-three, or 24.9 per cent., were poisoned.

Poisoning occurred among fourteen persons ingesting as little as 0.1 mg. of lead daily, over an average period of eight and one-quarter years.

The incidence of poisoning was distinctly lower in children under 10 years of age than among children from 10 to 20 years old or among adults, and was greater among adults than among children under 20.

The duration of exposure, except for very short periods, was not significant in its relation to the incidence of poisoning.

The incidence of poisoning was quite uniform among those ingesting varying amounts less than 1.5 mg. daily but was much greater as this amount was exceeded.

Under almost all conditions of comparison—by age, duration of exposure, amounts of lead ingested daily, and total amounts of lead ingested—women showed a lower incidence of poisoning than did men.

There is abundant reason for grateful acknowledgment of aid rendered in this study by the Massachusetts Department of Public Health, through the kindness of the late Commissioner, Dr. Eugene R. Kelly, particularly by Mr. A. R. G. Booth of the Water Laboratory; by the Harvard School of Public Health; by Mrs. T. M. Barber, formerly of the Industrial Clinic of the Massachusetts General Hospital; and by the Statistical Division of the Metropolitan Life Insurance Company.

BOOK REVIEWS

STAUBLUNGE UND STAUBLUNGENTUBERKULOSE. By Dr. *Franz Ickert*, Regierungs- und Medizinalrat in Gumbinnen; ehemalig. Leiter der Tuberkulose-Fürsorgestelle in Mansfeld. Die Tuberkulose und ihre Grenzgebiete in Einzeldarstellungen, Beihefte zu den Beiträgen zur Klinik der Tuberkulose und spezifischen Tuberkuloseforschung, edited by L. Brauer, Hamburg, and H. Ulrici, Sommerfeld, Vol. 4. Paper. Pp. 64 with illustrations, bibliography, and index. Berlin: Julius Springer, 1928.

This is a brief general review of the entire subject covering both the experimental and the clinical aspects of the dust problem. It concludes with a thorough bibliography covering the more recent literature. The author's summary is as follows:

On the one hand some authors consider that every worker in dust must anticipate dust damage to his lungs; others again consider the danger from dust as of so little significance that they do not care to regulate the purity of the air in workrooms; and still others advocate the exposure of cases of pulmonary disease to dusty atmospheres in order to promote healing. In the face of such contradictions the truth lies usually in a mid-position and one turns to the special case and single groups of surroundings.

From all of the known material which has been covered, little can be gathered with entire safety. Reasonably safe conclusions seem to be as follows:

1. With regard to the origin of dust injury and injury plus tuberculosis, the danger from the dust seems to grade downward from crystalline silica (SiO_2) as the worst offender. Next to crystalline silica stands emery (SiC); metallic dust is also very dangerous.

2. Infection, especially tuberculosis, plays an important rôle in the origin of dust damage, and tuberculosis is the essential agent in promoting the further development and spread of the pneumonokoniosis.

3. The pulmonary tuberculosis of dust disease has a predominantly indurative (cirrhotic) character. It is characterized by a long course, is a prominent cause of death for old persons, and is exceptional in other ways, for example, in the peculiar effects upon the constitution of the dust worker, and, finally, in the amount of infection found among relatives. One may correctly enough speak of an especial form of pulmonary tuberculosis, namely, "Staublungentuberkulose" (literally, dusted-lung tuberculosis). The course of the disease will be determined by the resistance of the individual, the type of dust, and other conditions, for example, the social status of the patient.

From these premises the prevention of dust damage follows logically:

1. Through efficient removal of dust, especially dust containing silica.

2. Through exclusion of tuberculous persons from dusty work in order to protect healthy workmen from infection and to keep tuberculous patients clear of dust damage. These ends are attainable by careful selection for employment and close oversight of the workmen. In South Africa, the operation of such supervision is known to be effective.

It is unfortunate that in Germany dust damage to the lungs and the same condition plus tuberculosis are not classed as industrial diseases as they are in other lands. Such recognition, after the English example, is desirable.

Investigations are still very necessary. The researches can best be carried out at a central point. The institute of Watkins-Pitchford in South Africa serves as a good example for such centralization of activity.

—*C. K. Drinker.*

THE ULTRA-VIOLET RAYS: THEIR ACTION ON INTERNAL AND NERVOUS DISEASES AND USE IN PREVENTING LOSS OF COLOR AND FALLING OF THE HAIR. By *Arnold Lorand*, M.D. (Vienna), Physician at the Carlsbad Springs, Czecho-Slovakia; Author of "Old Age Deferred," "Health through Rational Diet," etc. Cloth. Pp. 258 with index. Philadelphia: F. A. Davis Co., 1928.

The tremendous vogue of light therapy in recent years has inspired naturally enough a great deal of literature, mostly of a popular nature. Dr. Lorand's book on the subject comes within this class of popular treatise—with perhaps more emphasis on some phases of the subject than is accorded in the usual literature. The following divisions will give some idea of its contents: Part I. The Healing Properties of Natural and Artificial Sunlight; Part II. The Treatment of Falling Hair by the Ultra-Violet Rays; Part III. The Treatment by the Quartz Light of Graying Hair.

One will look in vain for data of scientific value, nor is the discussion at all critical. The material presented is based mostly upon clinical observation. None the less the book has the merit of being extremely readable and the author presents his viewpoint with a freshness that is a pleasant relief to the dreariness of many treatises upon ultraviolet radiation.—*L. T. Fairhall.*

BOOKS RECEIVED

Books received are acknowledged in this column, and such acknowledgment must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers and as space permits.

HAY-FEVER AND ASTHMA. THEIR CAUSE, PREVENTION AND TREATMENT. By *Ray M. Balyeat*, M.A., M.D., F.A.C.P., Instructor in Medicine and Lecturer on Allergic Diseases in the University of Oklahoma Medical School; Consulting Physician to St. Anthony's Hospital, and to the State University Hospital; Member of the American Association for the Study of Allergy; Vice-President, Oklahoma Academy of Science; Director of the Balyeat Hay-Fever and Asthma Clinic, Oklahoma City. Second edition. Cloth. Pp. 310 with illustrations and index. Philadelphia: F. A. Davis Co., 1928.

INTERNATIONAL CLINICS. Volume I. Thirty-Eighth Series, 1928. Cloth. Pp. 307 with index. Philadelphia and London: J. B. Lippincott Co., 1928.

J. I. H.
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